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Diarrhea induced by high doses of nicotinamide in dialysis patients

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To the Editor: Takahashi *et al.*[1] have recently described the effect of nicotinamide for controlling hyperphosphatemia in dialysis patients. However, Rottembourg *et al.*[2] reported thrombocytopenia in the same setting. We have started a prospective, open-label trial to study the efficiency and safety of nicotinamide in our hemodialysis patients with resistant hyperphosphatemia. Unlike Takahashi, we did not stop other phosphate binders. Six patients have already been included. Two patients were intolerant to sevelamer and thus only treated with calcium carbonate. The other four patients received a combined therapy. Five patients developed diarrhea during the study. In two of them, it was so important that nicotinamide had to be stopped. After discontinuing nicotinamide, diarrhea disappeared. In the three other patients, diarrhea improved with decreasing the dose of nicotinamide. The diarrhea started at a mean dose of 1050 ± 447 mg. We observed a significant decrease in phosphatemia while on treatment (from 85 ± 8.7 to 54 ± 12.1 mg/l; $P = 0.0048$ for the repeated measure analysis of variance), but no effect on platelet count (from 260.167 ± 40.435 to 281.167 ± 51.503; $P = 0.33$).

Diarrhea induced by high doses of nicotinamide is not described in non-renal failure population.[3] However, in Takahashi's study, 7.8% of the patients developed such an adverse effect.[1] In our population, diarrhea induced by nicotinamide might also be related to the simultaneous use of calcium carbonate and/or sevelamer. If nicotinamide proves to be useful in the management of hyperphosphatemia, more studies are needed to evaluate its safety at high doses.

We have no conflict of interest to declare.

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2. Rottembourg JB, Launay-Vacher V, Massard J. Thrombocytopenia induced by nicotinamide in hemodialysis patients. *Kidney Int* 2005; **68**: 2911–2912.
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Tamm–Horsfall protein knockout mice do not develop medullary cystic kidney disease

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To the Editor: Medullary cystic kidney disease type 2 (MCKD2) and familial juvenile hyperuricemic nephropathy (FJHN) are similar autosomal-dominant renal diseases with shared features, including polyuria, progressive renal failure, and abnormal urate handling, which leads to hyperuricemia and gout. Both diseases are associated with corticomedullary cysts, interstitial fibrosis secondary to infiltration by inflammatory cells, and marked thickening of tubular basement membranes.[1] Recent genetic studies in humans have shown mutations in the *UMOD* (uromodulin, Tamm–Horsfall, THP) gene in patients with MCKD2 and FJHN.[2] It has been suggested that MCKD2 and FJHN might represent different forms of the same disease. Patients with the MCKD/FJHN complex have also been shown to have reduced excretion of THP in urine.[3] It has remained unclear if changes of MCKD/

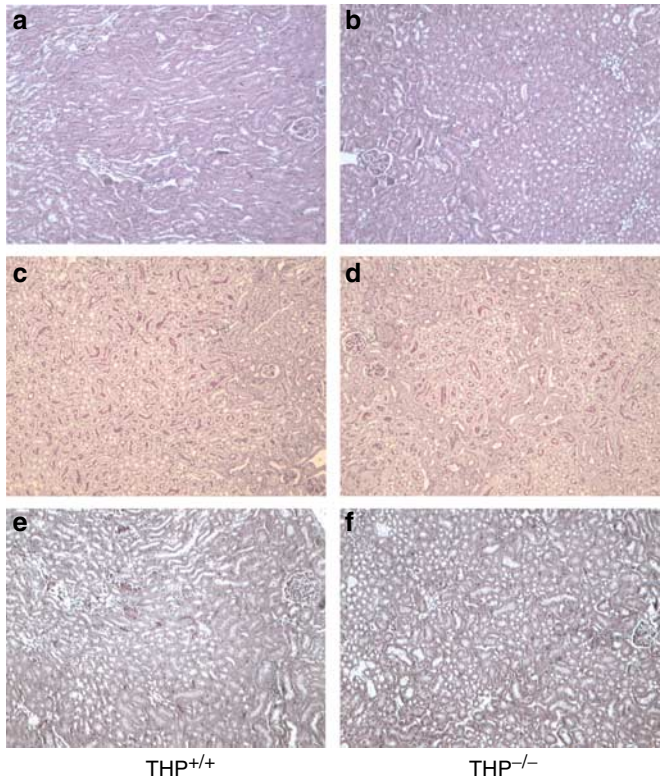

Figure 1 | Renal Histology. (**a** and **b**) Kidney sections from THP + / + and THP − / − mice stained with hematoxylin and eosin, (**c** and **d**) periodic acid schiff , and with (**e** and **f**) Masson's trichrome (original magnification × 100). Renal histology was normal in all mice. There were no areas of fibrosis or cystic change in the renal cortex or medulla from either group of mice.